

# FECAL METALS



**LAB#: F050929-0488-1**  
**PATIENT: Fen Hui Chen**  
**SEX: Female**  
**AGE: 54**

**CLIENT#: 28423**  
**DOCTOR: Fen-Hui Chen, DDS**

**1030 Pearl St #6**  
**La Jolla, CA 92037**

## POTENTIALLY TOXIC METALS

METALS	RESULT mg/kg	REFERENCE RANGE	PERCENTILE	
			68 <sup>th</sup>	95 <sup>th</sup>
Mercury	0.201	<.05 w/o amalgams*		
Mercury	0.201	<.5 with amalgams*		
Antimony	0.048	< 0.080		
Arsenic	1.21	< 0.30		
Beryllium	< dl	< 0.009		
Bismuth	0.027	< 0.050		
Cadmium	0.60	< 0.50		
Copper	30	< 60		
Lead	0.22	< 0.50		
Nickel	4.9	< 8.0		
Platinum	< dl	< 0.003		
Thallium	0.016	< 0.020		
Tungsten	0.029	< 0.090		
Uranium	0.117	< 0.120		

## % WATER CONTENT

	RESULT % H <sub>2</sub> O	EXPECTED RANGE	MEAN			
			2SD LOW	1SD LOW	72.5%	1SD HIGH
% WATER CONTENT	73.9	60-85%				

## DISCUSSION

Analysis of elements in feces provides a comprehensive evaluation of environmental exposure, accumulation and endogenous detoxification of potentially toxic metals. For several toxic elements such as mercury, cadmium, lead, antimony and uranium, biliary excretion of metals into feces is the primary natural route of elimination from the body. Studies performed at DDI demonstrate that the fecal mercury content and number of amalgam surfaces are highly correlated, as is the case for post-DMPS urine mercury levels and amalgam surface area.

Results are reported as mg/kg dry weight of feces to eliminate the influence of variability in water content of fecal specimens. The reference values that appear in this report have been derived from both published data and in-house studies at DDI. \*Due to exposure to mercury in the oral cavity, people with dental amalgams typically have a considerably higher level of mercury in the feces than individuals without dental amalgams; therefore, two reference ranges have been established for mercury.

To provide guidance in interpretation of results, patient values are plotted graphically with respect to percentile distribution of the population base. Since this test reflects both biliary excretion and exposure (metals to which the patient is exposed may not be absorbed), it may not correlate with overt clinical effects. Further testing can assist in determining whether the metals are from endogenous (biliary excretion) or exogenous (oral exposure) sources.

1. Bjorkman, L, Sandborgh-Englund, G, and Ekstand, J., Mercury in Saliva and Feces after Removal of Amalgam Fillings. Toxicology & Applied Pharmacology 144: 156-162 (1997)
2. Zalups, R, Progressive Losses of Renal Mass and the Renal and Hepatic Disposition of Administered Inorganic Mercury. Toxicology & Applied Pharmacology 130: 121-131 (1995)
3. Adamsson, E., Piscator, M., and Nogawa, K., Pulmonary and Gastrointestinal Exposure to Cadmium Oxide Dust in a Battery Factory. Environmental Health Perspectives, 28: 219-222 (1979)
4. Smith, J., et al., The Kinetics of Intravenously Administered Methyl Mercury in Man. Toxicology & Applied Pharmacology 128:251-256 (1994)
5. Bass, D., et al., "Measurement of Mercury in Feces", Poster presentation 1999 AACC

## SPECIMEN DATA

Comments:

Date Collected:	Provocation:	Dental Amalgams: <b>not indicated</b>
Date Received: 9/29/2005	Detoxification Agent:	Quantity:
Date Completed: 10/6/2005	Dosage:	Methodology: ICP-MS v02.00

## MERCURY HIGH

### FecalHG

Mercury (Hg) is an extremely toxic element. Fecal Hg is an excellent measure of exposure and possible accumulation of the element. Both fecal and urinary excretion are the main elimination routes for inorganic and methyl mercury.

It is quite clear that sensitivity to Hg varies greatly among individuals; some individuals exhibit extreme symptoms with levels of Hg which are without obvious effects in others. The symptomatology of Hg excess can depend on many factors: the chemical form of absorbed Hg and its transport in body tissues, presence of other synergistic toxics (Pb and Cd have such effects), presence of disease that depletes or inactivates lymphocytes or is immunosuppressive, organ levels of xenobiotic chemicals and sulfhydryl-bearing metabolites (e.g. glutathione), and the concentration of protective nutrients, (e.g. zinc, selenium, vitamin E). Early signs of mercury contamination include: decreased senses of touch, hearing, vision and taste, metallic taste in the mouth, fatigue or lack of physical endurance, and increased salivation. Symptoms may progress with moderate or chronic exposure to include: anxiety, depression, anorexia, numbness and paresthesias, headaches, hypertension, irritability and excitability, and immune suppression, possibly immune dysregulation. Advanced disease processes from mercury toxicity include: tremors and incoordination, anemia, psychoses, manic behaviors, possibly autoimmune disorders, renal dysfunction or failure.

Mercury is commonly used in: dental amalgams (50% by weight), explosive detonators, in elemental or liquid form for thermometers, barometers, and laboratory equipment; batteries and electrodes, some vaccines and in fungicides and pesticides. The fungicide and pesticide use of mercury (including that in paints) has declined due to environmental concerns, but mercury residues persist from past use. Methylmercury, the common, most poisonous form, occurs by methylation in aquatic biota or sediments, both freshwater and ocean sediments. Methylmercury accumulates in aquatic animals and fish and is concentrated up the food chain reaching high concentrations in large fish and predatory birds. Except for fish, the human intake of dietary mercury is negligible unless food is contaminated with one of the previously listed forms/sources.

Data collected at DDI indicate positive correlations between fecal Hg levels and the number of amalgams, and the amount of fish consumed.

Hg burden can be confirmed by urine elements analysis. Comparison of urine Hg levels pre and post provocation (DMPS, DMSA, D-penicillamine) permit differentiation between recent uptake and retention in the body.

## ARSENIC HIGH

### FecalAS

Fecal Arsenic (As) can be used as an estimate of exposure to the element. Inorganic As accumulates in hair, nails, skin, thyroid gland, bone and the gastrointestinal tract. Organic As is rapidly and mainly excreted in the urine and a smaller percentage in the feces.

As can cause malaise, muscle weakness, vomiting, diarrhea, dermatitis, and skin cancer. Long-term exposure may affect the peripheral nervous, cardiovascular and hematopoietic systems. As

is a major biological antagonist to selenium. Arsenic, along with antimony is often found in fecal specimens from autistic children.

Common sources of As are insecticides (calcium and lead arsenate), fungicides (orchards, vineyards), well water, smog, shellfish (arsenobetaine), and industrial exposure, particularly in the manufacture of electronic components (gallium arsenide), wood preservatives (pressure treated wood/sawdust).

As burden can be confirmed by urine elements analysis. Comparison of urine As levels pre and post provocation (DMPS, DMSA, D-penicillamine) permit differentiation between recent uptake and retention in the body.